

045106

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SEARCH REQUEST FORM

Scientific and Technical Information Center

Requester's Full Name: JANE ZARA Examiner #: 77512 Date: 6/28/01
 Art Unit: 1635 Phone Number 306-5820 Serial Number: 09/684/061
 Mail Box and Bldg/Room Location: 11003 Results Format Preferred (circle) PAPER DISK E-MAIL

11E12
 If more than one search is submitted, please prioritize searches in order of need.

 Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc, if known. Please attach a copy of the cover sheet, pertinent claims, and abstract.

Title of Invention: AS Comp's & Cancer Treat. Method

Inventors (please provide full names): BARTHELMEZ et al.

Earliest Priority Filing Date: 10/6/00

For Sequence Searches Only Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.

Please Search Seq ID

No 1.

Please limit to 60 NT'S.

THANKS

Point of Contact:
 Mona Smith
 Technical Info. Specialist
 CM1 12C14 Tel: 308-3278

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Date Searcher Picked Up: 7/2/01

Date Completed: 7/5/01

Searcher Prep & Review Time: 5

Clerical Prep Time: _____

Online Time: 7

Type of Search

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AA Sequence (#) _____

Structure (#) _____

Bibliographic _____

Litigation _____

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Patent Family _____

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Dr.Link _____

Lexis/Nexis _____

Sequence Systems _____

WWW/Internet _____

Other (specify) _____

7/2/01 /A B S 203
 7/3 C. 12

09/684,061

Set	Items	Description
S1	0	STEM (S) INFUS\$ (S) CIRCULA\$
S2	0	(EX ADJ VIVO) (S) STEM
S3	3	(EX VIVO) (S) STEM
S4	3	RD (unique items)

>>>KWIC option is not available in file(s): 399

4/3,K/1 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2002 Inst for Sci Info. All rts. reserv.

09855075 Genuine Article#: 457XL No. References: 65
Title: Stem cell factor: Biology and relevance to clinical practice
Author(s): Smith MA; Pallister CJ; Smith JG (REPRINT)
Corporate Source: Royal United Hosp NHS Trust, Dept Haematol, Combe Pk/Bath
BA1 3NG/Avon/England/ (REPRINT); Royal United Hosp NHS Trust, Dept
Haematol, Bath BA1 3NG/Avon/England/; Univ W England, Ctr Biomed
Res, Bristol BS16 1QY/Avon/England/
Journal: ACTA HAEMATOLOGICA, 2001, V105, N3, P143-150
ISSN: 0001-5792 Publication date: 20010000
Publisher: KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

4/3,K/2 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2002 Inst for Sci Info. All rts. reserv.

07586050 Genuine Article#: 185BR No. References: 42
Title: Bone morphogenetic proteins regulate the developmental program of human hematopoietic stem cells
Author(s): Bhatia M (REPRINT) ; Bonnet D; Wu DM; Murdoch B; Wrana J; Gallacher L; Dick JE
Corporate Source: JOHN P ROBERTS RES INST, DEPT GENE THERAPY & MOL VIROL, 100 PERTH DR/LONDON/ON N6A 5K8/CANADA/ (REPRINT); UNIV WESTERN ONTARIO, DEPT MICROBIOL & IMMUNOL/LONDON/ON N6A 5K8/CANADA/; HOSP SICK CHILDREN, PROGRAM CANC BLOOD/TORONTO/ON M5G 1X8/CANADA/; HOSP SICK CHILDREN, RES INST, PROGRAM DEV BIOL/TORONTO/ON M5G 1X8/CANADA/; UNIV TORONTO, DEPT MOL & MED GENET/TORONTO/ON M5G 1X8/CANADA/
Journal: JOURNAL OF EXPERIMENTAL MEDICINE, 1999, V189, N7 (APR 5), P 1139-1147
ISSN: 0022-1007 Publication date: 19990405
Publisher: ROCKEFELLER UNIV PRESS, 1114 FIRST AVE, 4TH FL, NEW YORK, NY 10021
Language: English Document Type: ARTICLE (ABSTRACT AVAILABLE)

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DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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03640496 Genuine Article#: PU149 No. References: 19
Title: PRODUCTION OF FUNCTIONAL MYELOID CELLS FROM CD34-SELECTED HEMATOPOIETIC PROGENITOR CELLS USING A CLINICALLY RELEVANT EX-VIVO EXPANSION SYSTEM
Author(s): LILL MC; LYNCH M; FRASER JK; CHUNG GY; SCHILLER G; GLASPY JA; SOUZA L; BALDWIN GC; GASSON JC
Corporate Source: UNIV CALIF LOS ANGELES, SCH MED, DEPT MED, DIV HEMATOL ONCOL, 11-934 FACTOR/LOS ANGELES//CA/90024; UNIV CALIF LOS ANGELES, SCH MED, DEPT MED, DIV HEMATOL ONCOL/LOS ANGELES//CA/90024; JONSSON COMPREHENS CANC CTR/LOS ANGELES//CA/00000; AMGEN CORP/THOUSAND OAKS//CA/91320; UNIV CALIF LOS ANGELES, SCH MED, DEPT BIOL CHEM/LOS ANGELES//CA/90024; UNIV CALIF LOS ANGELES, SCH MED, INST MOLEC BIOL/LOS ANGELES//CA/90024
Journal: STEM CELLS, 1994, V12, N6 (NOV), P626-637
ISSN: 1066-5099

Language: ENGLISH Document Type: ARTICLE (Abstract Available)

Set	Items	Description
S1	0	STEM (S) INFUS\$ (S) CIRCULA\$
S2	0	(EX ADJ VIVO) (S) STEM
S3	3	(EX VIVO) (S) STEM
S4	3	RD (unique items)
S5	1	(EX VIVO) (S) ANTISENSE

>>>KWIC option is not available in file(s): 399

5/3,K/1 (Item 1 from file: 34)
 DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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07618466 Genuine Article#: 188DG No. References: 35
**Title: Antisense oligonucleotides: local delivery enhances their
 therapeutic potential**
 Author(s): Nyce JW (REPRINT)
 Corporate Source: EPIGENESIS PHARMACEUT INC,DEPT MOL PHARMACOL &
 THERAPEUT/PRINCETON//NJ/08543 (REPRINT)
 Journal: EXPERT OPINION ON THERAPEUTIC PATENTS, 1999, V9, N3 (MAR), P
 263-267
 ISSN: 1354-3776 Publication date: 19990300
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 LONDON N6 5QJ, ENGLAND
 Language: English Document Type: REVIEW (ABSTRACT AVAILABLE)
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Rm 300. E9

[0093] The foregoing methods and compositions also are useful for ex vivo expansion of stem cells after transfection with retroviral or other vectors containing a heterologous nucleic acid (e.g., an antisense oligonucleotide, a nucleic acid encoding a therapeutic protein or peptide) for gene therapy applications. Stem cells into which a heterologous nucleic acid has been introduced ex vivo can be introduced into the subject using the known methods for implanting transfected cells into a human for gene therapy. See, e.g., U.S. Pat. No. 5,399,346 ("Gene Therapy") issued to Anderson et al.; PCT International application no. PCT/US92/01890 (Publication No. WO 92/15676, "Somatic Cell Gene Therapy", claiming priority to U.S. Ser. No. 667,169, filed Mar. 8, 1991, inventor I. M. Verma); PCT International application no. PCT/US89/05575 (Publication No. WO 90/06997, "Genetically Engineered Endothelial Cells and Use Thereof", claiming priority to U.S. Ser. No. 283,586, filed Dec. 8, 1989, inventors Anderson, W. F. et al.).

6,068,836

5,665,350

6,255,112

6,258,597

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Set	Items	Description
S1	180	EVI(3N)ZINC(W)FINGER
S2	5	S1(S)(ANTISENSE OR RIBOZYME?)
S3	5	RD (unique items)

01146446 97611956

Molecular mechanism of blastic crisis in chronic myelocytic leukemia.
(Meeting abstract).

Mitani K

Third Department of Internal Medicine, Faculty of Medicine, University of
Tokyo, Bunkyo ku, Tokyo 113, Japan
Non-serial; Leukemia and Lymphoma, Pathogenesis and Treatment, Molecular
Aspects, p. 37. 18th Symposium of the International Association for
Competitive Research on Leukemia and Related Diseases, Kyoto, Japan,
October 29-November 3, 1995.: 1995

Languages: ENGLISH

Document Type: MEETING ABSTRACTS

... EVI-1 fusion protein of 180 kD containing amino-terminal half of AML1
including a runt homology domain which is fused to the entire of *zinc*
finger *EVI* -1 protein. Thus, AML1/EVI-1 fusion protein is a chimeric
transcription factor including a runt homology domain from AML1 and two
zinc *finger* domains from *EVI*-1, totally three DNA binding domains,
and an acidic domain from EVI-1. To evaluate the effect of the AML1/EVI-1
fusion protein on cell growth of SKH1 cells, we prepared the synthetic
antisense oligonucleotides with 18 nucleotides spanning the junction
point between AML1 and EVI-1 sequences and those with 4 point mutations in
their sequences as a negative control. The *antisense* oligonucleotides
suppressed 3H-thymidine incorporation in SKH1 cells and decreased the cell
number of the cells in comparison with those including 4 point mutations,
suggesting...

...AML1/EVI-1 into Rat1 clones harboring BCR/ABL conferred enhanced ability
for anchorage independent growth. The analysis using deletion mutants
showed that the second *zinc* *finger* domain within the *EVI*-1 was the
functional region critical for transformation. The AML1/EVI-1 stimulated
AP-1 activity through the TRE site as in the EVI-1...

3/3,K/4 (Item 2 from file: 159)

DIALOG(R)File 159:Cancerlit

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01061406 96604994

Oncogenic potentials of the AML-1/EVI-1 fusion protein derived from the
t(3;21) (q26;q22) translocation in blastic crisis of chronic myelocytic
leukemia (Meeting abstract).

Mitani K; Kurokawa M; Ogawa S; Tanaka T; Yazaki Y; Hirai H

Third Dept. of Internal Medicine, Faculty of Medicine, Univ. of Tokyo,
Tokyo, Japan

Blood; 84(10, Suppl 1):229a 1994 ISSN 0903-1936

Languages: ENGLISH

Document Type: JOURNAL ARTICLE

... 1 fusion protein of 180 kD containing amino-terminal half of AML-1
including a runt homology domain which is fused to the entire of *zinc*
finger *EVI* -1 protein (Mitani K et al, EMBO J; 13:504 1994). Thus
AML-1/EVI-1 fusion protein is a chimeric transcription factor including a
runt homology domain from AML-1 and two *zinc* *finger* domains from *EVI*
-1, totally three DNA binding domains, and an acidic domain from EVI-1 as a
transcriptional activation domain. To evaluate the effects of the
AML-1/EVI-1 fusion protein on cell growth of SKH-1 cells, we prepared the
synthetic *antisense* oligonucleotides with 16 nucleotides spanning the
junction point between AML-1 and EVI-1 sequences and those with 4 point
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point mutations, suggesting...

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anchorage independent growth. The analysis using deletion mutants showed
that the second *zinc* *finger* domain within the *EVI* -1 was the
functional region critical for transformation. All these data suggest that

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the AML-1/EVI-1 could play an important role in leukemic...

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3/3,K/5 (Item 3 from file: 159)
DIALOG(R) File 159: Cancerlit
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00987458 95699358

Generation of the AML1/EVI-1 fusion gene in the t(3;21)(q26;q22) translocation causes blastic crisis of chronic myelocytic leukemia (Meeting abstract).

Mitani K; Ogawa S; Miyoshi H; Mano H; Yazaki Y; Ohki M; Hirai H
Univ. of Tokyo, Tokyo, Japan
Non-serial; Molecular Biology of Hematopoiesis; 8th Symposium. July 9-13, 1993, Basel, Switzerland, p. 79, 1993.: 1993
Languages: ENGLISH
Document Type: JOURNAL ARTICLE

... cells carrying t(3;21). The fusion protein contains amino-terminal half of AML1 including a runt homology domain which is fused to the entire *zinc* *finger* *EVI* -1 protein. The AML1/EVI-1 fusion has been demonstrated to be consistent among all three cases of the t(3;21)-carrying leukemia. Synthetic *antisense* oligonucleotides with 20 nucleotides spanning the initiation sites of AML1 or EVI-1 sequences suppress 3H-thymidine incorporation in SKH1 cells, suggesting that the AML1...
?

ILL

Set	Items	Description
S1	180	EVI(3N)ZINC(W)FINGER
S2	5	S1(S)(ANTISENSE OR RIBOZYME?)
S3	5	RD (unique items)

>>>KWIC option is not available in file(s): 41, 77, 399

3/3,K/1 (Item 1 from file: 94)
DIALOG(R)File 94:JICST-EPlus
(c)2001 Japan Science and Tech Corp(JST). All rts. reserv.

02664091 JICST ACCESSION NUMBER: 96A0132126 FILE SEGMENT: JICST-E
Analysis of molecular mechanism in blastic crisis of chronic myelocytic leukemia.

MITANI KINUKO (1)
(1) Univ. of Tokyo, Fac. of Med.
Nissan Kagaku Shinko Zaidan Kenkyu Hokokusho(Research Projects in Review,
Nissan Science Foundation), 1995, VOL.18(1995), PAGE.235-238, REF.6
JOURNAL NUMBER: X0726AAW ISSN NO: 0911-4572
UNIVERSAL DECIMAL CLASSIFICATION: 616-006-09
LANGUAGE: Japanese COUNTRY OF PUBLICATION: Japan
DOCUMENT TYPE: Journal
ARTICLE TYPE: Short Communication
MEDIA TYPE: Printed Publication

...ABSTRACT: AML1/EVI-1 fusion protein of 180kD containing amino-terminal half of AML1 including a runt homology domain which is fused to the entire of *zinc* *finger* *EVI*-1 protein. Thus, the AML1/EVI-1 fusion protein is a chimeric transcription factor including a runt homology domain from AML1 and two *zinc* *finger* domains from *EVI*-1, totally three DNA binding domains, and an acidic domain from EVI-1. To evaluate the effect of the AML1/EVI-1 fusion protein on cell growth of SKH1 cells, we prepared the synthetic *antisense* oligonucleotides with 18 nucleotides spanning the junction point between AML1 and EVI-1 sequences and those with 4 point mutations in their sequences as a negative control. The *antisense* oligonucleotides suppressed 3H-thymidine incorporation in SKH1 cells and decreased the cell number of the cells in comparison with those including 4 point mutations, suggesting...

...AML1/EVI-1 into Rat1 clones harboring BCR/ABL conferred enhanced ability for anchorage independent growth. The analysis using deletion mutants showed that the second *zinc* *finger* domain within the *EVI*-1 was the functional region critical for transformation. The AML1/EVI-1 could stimulate AP-1 activity through the TRE site as in the EVI...

3/3,K/2 (Item 1 from file: 399)
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134294085 CA: 134(21)294085d PATENT
Hematopoietic stem cells (HSC) treated with antisense oligonucleotide targeted to genes preferentially expressed in HSC and cancer treatment
INVENTOR(AUTHOR): Bartelmez, Stephen H.; Iversen, Patrick L.
LOCATION: USA
ASSIGNEE: Avi Biopharma, Inc.
PATENT: PCT International ; WO 0125422 A2 DATE: 20010412
APPLICATION: WO 2000US27636 (20001006) *US PV158340 (19991007)
PAGES: 36 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/11A;
A61K-031/712B; C12N-005/10B DESIGNATED COUNTRIES: AU; CA; JP; KR
DESIGNATED REGIONAL: AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT;
LU; MC; NL; PT; SE

3/3,K/3 (Item 1 from file: 159)
DIALOG(R)File 159:Cancerlit
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01146446 97611956

**Molecular mechanism of blastic crisis in chronic myelocytic leukemia.
(Meeting abstract).**

Mitani K

Third Department of Internal Medicine, Faculty of Medicine, University of Tokyo, Bunkyo ku, Tokyo 113, Japan

Non-serial; Leukemia and Lymphoma, Pathogenesis and Treatment, Molecular Aspects, p. 37. 18th Symposium of the International Association for Competitive Research on Leukemia and Related Diseases, Kyoto, Japan, October 29-November 3, 1995.: 1995

Languages: ENGLISH

Document Type: MEETING ABSTRACTS

... EVI-1 fusion protein of 180 kD containing amino-terminal half of AML1 including a runt homology domain which is fused to the entire of *zinc* *finger* *EVI* -1 protein. Thus, AML1/EVI-1 fusion protein is a chimeric transcription factor including a runt homology domain from AML1 and two *zinc* *finger* domains from *EVI*-1, totally three DNA binding domains, and an acidic domain from EVI-1. To evaluate the effect of the AML1/EVI-1 fusion protein on cell growth of SKH1 cells, we prepared the synthetic *antisense* oligonucleotides with 18 nucleotides spanning the junction point between AML1 and EVI-1 sequences and those with 4 point mutations in their sequences as a negative control. The *antisense* oligonucleotides suppressed 3H-thymidine incorporation in SKH1 cells and decreased the cell number of the cells in comparison with those including 4 point mutations, suggesting...

...AML1/EVI-1 into Rat1 clones harboring BCR/ABL conferred enhanced ability for anchorage independent growth. The analysis using deletion mutants showed that the second *zinc* *finger* domain within the *EVI*-1 was the functional region critical for transformation. The AML1/EVI-1 stimulated AP-1 activity through the TRE site as in the EVI-1...

3/3,K/4 (Item 2 from file: 159)

DIALOG(R) File 159:Cancerlit

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01061406 96604994

Oncogenic potentials of the AML-1/EVI-1 fusion protein derived from the t(3;21) (q26;q22) translocation in blastic crisis of chronic myelocytic leukemia (Meeting abstract).

Mitani K; Kurokawa M; Ogawa S; Tanaka T; Yazaki Y; Hirai H

Third Dept. of Internal Medicine, Faculty of Medicine, Univ. of Tokyo, Tokyo, Japan

Blood; 84(10, Suppl 1):229a 1994 ISSN 0903-1936

Languages: ENGLISH

Document Type: JOURNAL ARTICLE

... 1 fusion protein of 180 kD containing amino-terminal half of AML-1 including a runt homology domain which is fused to the entire of *zinc* *finger* *EVI* -1 protein (Mitani K et al, EMBO J; 13:504 1994). Thus AML-1/EVI-1 fusion protein is a chimeric transcription factor including a runt homology domain from AML-1 and two *zinc* *finger* domains from *EVI*-1, totally three DNA binding domains, and an acidic domain from EVI-1 as a transcriptional activation domain. To evaluate the effects of the AML-1/EVI-1 fusion protein on cell growth of SKH-1 cells, we prepared the synthetic *antisense* oligonucleotides with 16 nucleotides spanning the junction point between AML-1 and EVI-1 sequences and those with 4 point mutations in their sequences as a negative control. The *antisense* oligonucleotides suppressed 3H-thymidine incorporation in SKH1 cells and decreased the cell number of the cells in comparison with those including 4 point mutations, suggesting...

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the AML-1/EVI-1 could play an important role in leukemic...

3/3,K/5 (Item 3 from file: 159)

DIALOG(R)File 159:Cancerlit

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00987458 95699358

Generation of the AML1/EVI-1 fusion gene in the t(3;21)(q26;q22) translocation causes blastic crisis of chronic myelocytic leukemia (Meeting abstract).

Mitani K; Ogawa S; Miyoshi H; Mano H; Yazaki Y; Ohki M; Hirai H

Univ. of Tokyo, Tokyo, Japan

Non-serial; Molecular Biology of Hematopoiesis, 8th Symposium. July 9-13, 1993, Basel, Switzerland, p. 79, 1993.: 1993

Languages: ENGLISH

Document Type: JOURNAL ARTICLE

... cells carrying t(3;21). The fusion protein contains amino-terminal half of AML1 including a runt homology domain which is fused to the entire *zinc* *finger* *EVI* -1 protein. The AML1/EVI-1 fusion has been demonstrated to be consistent among all three cases of the t(3;21)-carrying leukemia. Synthetic *antisense* oligonucleotides with 20 nucleotides spanning the initiation sites of AML1 or EVI-1 sequences suppress 3H-thymidine incorporation in SKH1 cells, suggesting that the AML1...

?

ILL

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